on a profuse weakening diarrhœa. Afterward he used Merck's hepatic extract in half-gram to one gram tablets. These tablets had a decided diuretic effect and they increased the output of urea. There is only 0.066 gram of urea in one of Merck's tablets, and the elimination of urea is not, therefore, a mere throwing off of the actual tablet-content, but an effect of cell-stimulation. The food was weighed, and in one case for three days during which no hepatic extract was given the quantity of urine was 1320, 1675, and 1237 c.c.; on the next three days when one-half to one gram of the extract was given, it was 1700, 1995, and 1755 c.c. The output of urea for the first three days was 78.790 grams, for the second three days, 88.016 grams; on three following days, when two to four grams of the extract were given, the quantity of urine was 1580, 1650 and 1850 c.c.; and the total output of urea was 100.746 grams.

With an intention to improve the blood he gave fresh lamb-marrow, as had been suggested by Combe, Cervesato and Caccini after experiments made between 1895 and 1900, and the effect by inference appears to be good, although he does not describe it in detail.

REPORT OF A CASE OF PARATYPHOID FEVER.[1]

By John M. Swan, M.D.,
of Philadelphia.

The patient whose case forms the basis of this report was admitted to the Philadelphia General Hospital on June 23, 1905. It was thought at first that he was suffering from typhoid fever and that a consolidated area, which was made out at the apex of his right lung, was a patch of complicating bronchopneumonia. On the second day of his hospital residence a negative serum reaction with the bacillus typhosus was reported. Two days later a positive diazo reaction was demonstrated in his urine. A blood culture made at the same time that the serum reaction was studied was reported sterile. It was then supposed that the patient might be suffering from beginning tuberculosis on account of the consolidation at the right apex; but resolution gradually occurred in this area and the cough as well as the physical signs disappeared. On the eleventh day of his hospital residence agglutination reactions were attempted between the patient's blood serum and the alpha and beta paratyphoid organisms used by Fox[22] in his agglutination studies. In a 1:40 dilution there was what seemed to be a tendency to agglutination with the alpha organism at the end of one hour, but accuracy requires that the reaction be said to be negative. Six days later the patient was catheterized under proper aseptic precautions and the urine cultured in the Clinical Laboratory of the Hospital. A bacillus was obtained which was about the size of the bacillus typhosus. This organism was motile, non-gas producing, gave no liquefaction of gelatin, produced acid and did not coagulate milk. It clouded bouillon without the formation of a pellicle. The organism was agglutinated by the patient's blood, in dilutions of one to forty in twelve minutes. It was not affected by three known typhoid sera in the same dilution.. The diagnosis of paratyphoid fever was then considered confirmed.

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[1] Read at the meeting of the Section of General Medicine, College of Physicians, December 11, 1905.

At first the patient presented all the symptoms of typhoid fever, except enlargement of the spleen. He had suffered from diarrhœa, headache, fever and asthenia for two weeks and presented abundant rose spots. It was soon discovered, however, that his temperature was not following a typical course. Instead of the usual remittent temperature of typhoid fever, there were irregular fluctuations. In the first twenty-four hours it touched the normal point once. During the second twenty-four hours it was constantly at or above 110°, until just at the end of the period, when it dropped from 104° to normal after a chill. It then pursued a course which, together with the patch of consolidation, suggested tuberculosis. By the twelfth day of hospital residence the temperature had reached the normal point and it did not go above that during the remaining fourteen days that he was under observation. The pulse, as a rule, followed the temperature, except that it was weak after tub baths and after sponges. The respirations were somewhat more rapid than common.

During the first week there was a moderate acute nephritis as indicated by albuminuria with hyaline, blood, epithelial and pale granular casts. This condition had cleared by the eleventh day, when the urine was found normal.

The blood showed the low leukocyte count and moderate reduction of erythrocytes and of hæmoglobin usually found in typhoid fever.

The patient was discharged, cured, on the twenty-sixth day of hospital treatment.

The details of the case follow:

White, adult, male, aged thirty-five years, a native of Russia and by occupation a laborer, was admitted to the medical wards of the Philadelphia General Hospital on June 23, 1905, in the service of Dr. Stengel. He was complaining of diarrhœa, headache, fever and asthenia.

Family history. Unimportant.

Previous history. Unimportant.

Present illness began about two weeks before admission with malaise, fever, headache and diarrhœa. The patient is now constipated.

Examination. A well-developed, white, adult male. Skin and mucous membranes pale. Pupils equal, react to light, distance and convergence. Tongue heavily coated on dorsum; tip and edges red.

Chest. Chest fairly well developed, anteroposterior diameter sufficient. Intercostal angle obtuse; slight depression beneath right clavicle. Expansion not so marked at right apex as at left. Over the right apex there is impaired resonance, increased tactile fremitus, increased vocal resonance and whispering pectoriloquy. At both bases moist and crackling rales are heard. At the right base a pleural friction rub may be heard in addition. There is some cough and slight expectoration.

Heart. The apex beat is in its normal situation. Dulness extends from the third rib to the fifth interspace and from the midclavicular line to the right edge of the sternum. No adventitious sounds.

Abdomen. The abdomen is so distended and tympanitic that the liver dulness cannot be mapped out. The spleen is not palpable. There are no areas of tenderness. The skin of the abdomen presents an abundant crop of rose spots, which disappear on pressure.

June 24th. The patient's temperature is somewhat irregular and he does not stand his tubs well. The cough continues; but there is not enough expectoration to obtain a specimen for laboratory examination. The rales are still present. There is not much depression.

25th. Physical signs as yesterday. Serum reaction with bacillus typhosus negative. Pulse weak after tub.

26th. Abdomen somewhat distended. Spots persist. Stool greenish with yellowish-green curds. Respirations slightly increased. Cough persists. No expectoration.

27th. Diazo reaction positive. Sputum examined, but no tubercle bacilli were found. Blood culture made on June 25th, reported sterile. Pulse weak after a sponge.

28th. Cough ceased. Eruption disappearing. Abdomen tympanitic. Constipation with yellow, partly formed stools. Pulse somewhat stronger. Sponged ten minutes. Tongue coated on dorsum, red at tip and edges.

30th. Pulse more regular. Rales at bases still present. No splenic enlargement. Signs at right apex continue as at last note. Patient coughs when he sits up, rales still present, more marked at the left base. Pleural friction still present. Harsh breathing at both bases.

July 2d. The signs at the right apex have disappeared. The systolic sound at the apex of the heart is somewhat muffled. The first aortic sound is distant and weak and the same may be said of the first sound in the pulmonary area. Both aortic and pulmonic diastolic sounds are accentuated.

3d. Slight impairment of resonance at both bases with fine, moist rales. A few moist rales were heard in the left axilla which cleared up after coughing. Pleural friction rub at the base of the right lung. Muscular quality of the heart sounds decidedly deficient. No murmurs. Aortic diastolic sound decidedly accentuated. Liver dulness obscured by distended colon. Splenic dulness from the eighth to the twelfth ribs, edge not palpable.

4th. Blood was examined for agglutination with alpha and beta paratyphoid organisms (Fox), dilution 1:40. No evidence of agglutination with the beta organism. At the end of an hour there seemed to be a tendency of the alpha organism to agglutinate, but there was no loss of motion. Groups of one dozen or so organisms could be seen moving quite rapidly over rather limited areas. Preparation of the culture before the blood serum was added showed no such appearance.

7th. Ophthalmoscopic examination attempted but failed on account of the inability of the patient to understand the directions given.

10*th.* Since the last note the patient has been steadily improving. The signs in the lungs have entirely disappeared.

18*th.* Discharged cured.

Laboratory Reports from the Clinical Laboratory, Philadelphia General Hospital.

Urine. June 26th. Slight sediment; reaction, acid; specific gravity, 1016; no glucose; albumin present. Epithelial, granular, blood and hyaline casts. Epithelial cells.

27th. Slight sediment; reaction, acid; specific gravity, 1010; no glucose, albumin present. Diazo reaction positive. Hyaline casts and epithelial cells.

July 4th. Slight sediment; reaction, acid; specific gravity, 1025; no glucose, no albumin. Diazo reaction negative. Amorphous urates.

Blood. July 24th. Erythrocytes, 3,500,000; leukocytes, 7,000; hemoglobin, 80 per cent.

4th. Erythrocytes, 4,300,000; leukocytes, 6,400; hæmoglobin, 80 per cent.

Serum reaction with bacillus typhosus negative on June 24th and July 11th. Feces examined for bacillus typhosus on July 3d, with negative result. The bacillus coli communis was the most abundant organism present.

Blood culture sterile on June 25th.

On July 10th the patient was catheterized under aseptic precautions and one cubic centimeter of the urine obtained was plated. The following organism predominated.

Morphology. Identical with bacillus typhosus.

Motility. Actively motile.

Staining. Stains readily with all the ordinary stains and is decolorized by Gram's method.

Cultural Characteristics. Bouillon. A uniform turbidity. Agar stroke. Bluish-gray film. Gelatin stab. Growth follows the stab, no gas or liquefaction. Litmus agar. Acid production after seventy-two hours, remaining so until culture died. Litmus milk. No coagulation. Slight acid production lasting six days and then the reaction became alkaline and remained so. Glucose in sugar-free bouillon. No fermentation. Lactose and saccharose not fermented. Neutral red. Unchanged. Non-pathogenic for guinea-pigs. Indol production negative. Agglutination. Positive in 1:40 dilution in twelve minutes. The patient's blood repeatedly failed to agglutinate the bacillus typhosus. The blood from three typical cases of typhoid fever failed to agglutinate the organism obtained from the patient's urine.

Diagnosis. Paratyphoid fever complicated by bronchopneumonia at right apex; dry pleuritis at root of right lung and acute nephritis.

Since Achard and Bensaude[1] described the first cases of paratyphoid infection in 1896, one of which was diagnosed by a bacteriological examination of the urine and the other by a bacteriological examination of the pus from an infective arthritis of the sterno-clavicular joint, fifty-seven cases have been reported in the United States beside the one I report to-night.

Brill[2] reported seventeen cases in which the diagnosis was made on account of the persistent absence of the agglutination reaction with bacillus typhosus.

Gwyn[3] reported one case which was diagnosed by blood culture.

Strong[4] reported one case which was diagnosed by cultures made from the spleen postmortem.

Coleman and Buxton[5] reported one case which was diagnosed by blood culture.

Hewlett[6] reported one case which was diagnosed by blood culture.

Longcope[7] reported two cases which were diagnosed by blood culture. In one of these a similar organism was obtained postmortem from the heart's blood, lungs, liver and spleen.

Johnston[8] reported four cases, two of which were diagnosed by blood culture and two by the agglutination reaction between the patients' serum and known paratyphoid organisms.

Libman[9] reported one case which was diagnosed by blood culture, urine culture, and bacteriological examination of fluid aspirated from the gall-bladder.

Allen[10] reported three cases, one of which was diagnosed by culture of pus from an acute cholecystitis, one by blood culture and urine culture, and one by the agglutination test.

Pratt[11] reported three cases, one of which was diagnosed by culture of pus from a suppurative orchitis, one by bacteriological examination of fluid in the gall-bladder and the centres of calculi at operation for cholelithiasis and one by blood culture.

Jermain[12] reported three cases, one of which was diagnosed by agglutination, the other two by clinical methods.

Ruediger[13] reported two cases both of which were diagnosed by blood culture.

Henry[14] reported six cases which were diagnosed by agglutination tests.

Van Ingen and Frissell[15] reported four cases, one of which was diagnosed by bacteriological examination of the spleen postmortem, the other three by agglutination. Later Tuttle[16] reported the same cases, and added two more; one of which was diagnosed by agglutination and the other by clinical methods.

Wells and Scott[17] reported one case which was diagnosed by bacteriological examination of the organs postmortem.

T. H. Evans[18] reported three cases, one of which was diagnosed by absence of the agglutination reaction with bacillus typhosus.

G. Bernard Smith[20] reported one case which was diagnosed by blood culture.

James W. Walker[21] reported one case which was diagnosed by blood culture.

Of these cases, twenty-three were diagnosed by clinical methods; by which I mean that if a positive agglutination reaction were not obtained with bacillus typhosus the case was considered to be one of paratyphoid fever. In fourteen the diagnosis was made after bacteriological examination of the patient's blood. In fourteen the diagnosis was made by a positive reaction between the patient's blood and a pure bouillon culture of a known paratyphoid organism. In three the diagnosis was made by bacteriological examination of the pus from a postfebrile lesion. In three the diagnosis was made by bacteriological examination of the organs after death.

In two cases a paratyphoid organism was obtained from the patient's urine by culture methods; but as in both cases a positive blood culture had also been made I give the credit for making the diagnosis to the latter method.

It appears to me that the following proposition may be said to cover the method of diagnosticating this acute infectious disease from typhoid fever. In a case of supposed typhoid fever in which a positive agglutination reaction between the patient's blood serum and the bacillus typhosus is not obtained on the first trial, try a similar reaction with a paratyphoid organism. If this fails, a blood culture should be made. If for any reason a blood culture is sterile, or if it is impossible to make such a culture, the patient should be catheterized under aseptic conditions and his urine examined by bacteriological methods. The paratyphoid organism may thus be obtained and the diagnosis confirmed.

Warren Coleman[19] draws the following conclusions from a study of paratyphoid fever: (1) That paratyphoid infections cannot be distinguished from typhoid fever except by the recovery from the blood of the bacillus concerned, and its proper identification. (2) That the present state of our knowledge makes it advisable to consider typhoid fever clinically as a disease which may be caused by several members of the typhoid colon group of bacilli. (3) That the term paratyphoid fever is not only unnecessary but misleading.

It seems to me that these conclusions are not justified. If we take such ground, how shall we look upon the labors of Louis and Gerhard who did such excellent work in the separation of typhoid fever from typhus, and what shall we say of those who have been instrumental in differentiating German measles from measles?

I wish to take this opportunity to thank Dr. Stengel for the privilege of making this report.

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